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Gene Section
Review

MIR133B (microRNA 133b)

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Abstract: Review on MIR133B, with data on DNA/RNA, on the protein encoded and where the gene is implicated.

Identity

Other names: MIRN133B, miRNA133B, mir-133b

HGNC (Hugo): MIR133B

Location: 6p12.2

Local order: Based on MapViewer, gene flanking MIR133B oriented from centromere to telomere on 6p12.2 are:
- MCM3: minichromosome maintenance complex component 3
- IL17F: interleukin 17F
- IL17A: interleukin 17A
- **MIR133B: microRNA 133B**
- MIR206: microRNA 206
- PKHD1: polycystic kidney and hepatic disease 1 (autosomal recessive).

DNA/RNA

Description

Homologues have been discovered in several other species including invertebrates, most of which has multiple miR133 family members. For example, the human genome encodes three miR133 genes: miR133A1, miR133A2 and miR133B on chromosomes 18, 20 and 6, respectively.

Transcription

MiR133B is specifically modestly expressed in the substantia nigra pars compacta as well as in GABAergic neurons of cortex and cerebellum. The beginning and the end of the pri-miR133B sequence are unknown.

Pre-miR133B
miRBase accession number: MI0000822.
Length: 119 nucleotides.
Sequence:
5'-
CCTCAGAAGAAAGATGCCCCCTGCTCTGGCTG
GTCAAACGGAACCAAGTCCGTCTTCCTGAGAG
GTTTGGTCCCCTTCAACCAGCTACAGCAGGGCT
GGCAATGCCCAGTCCTTGGAGA-3'

Mature miR133B
miRBase accession number: MIMAT0000770.
Length: 22 nucleotides.
Sequence:
5'-TTTGGTCCCCTTCAACCAGCTA-3'

Pseudogene

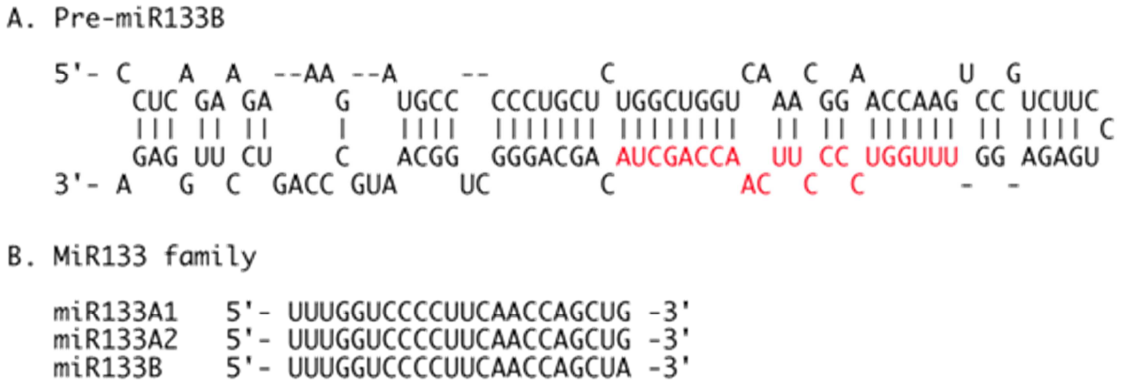
Pseudogenes were not reported.

Protein

Note
MicroRNAs are not translated into aminoacids.

Mutations

Note
A single nucleotide variation was reported (Reference SNP ID: rs112599381). The variation is T>C at position 52013754 (according to hg19-Feb_2009) with unknown frequency.



A. Homo sapiens stem-loop structure of pre-miR133B. Red characters indicate mature miR133B sequence. B. The human miR133 family members.

Implicated in

Bladder cancer

Note

A subset of 7 miRNAs (miR-145, miR-30a-3p, miR-133a, miR-133b, miR-195, miR-125b and miR-199a*) were found to be significantly downregulated in bladder cancers (Ichimi et al., 2009).

Prognosis

Dyrskjøt et al. identified several miRNAs with prognostic potential for predicting bladder tumor progression (e.g., miR-129, miR-133b, and miR-518c*) (Dyrskjøt et al., 2009).

miR-133a and miR-133b were found to inhibit cell proliferation, migration and invasion in T24 and EJ cells.

The first evidence was provided that miR-133a and miR-133b may directly target the epidermal growth factor receptor in bladder cancer (Zhou et al., 2012).

Cervical carcinoma

Note

Transfection with miR-133b rendered HeLa cells sensitive to TNF-a, TRAIL and FasL-induced cell death, by targeting the antiapoptotic protein Fas apoptosis inhibitory molecule (FAIM) (Patron et al., 2012).

Oncogenesis

miR-133b enhances cell proliferation and colony formation by targeting mammalian sterile 20-like kinase 2 (MST2), cell division control protein 42 homolog (CDC42) and ras homolog gene family member A (RHOA), which subsequently results in activation of the tumorigenic protein kinase B alpha (AKT1) and mitogen-activated protein kinase (ERK1 and ERK2, here abbreviated as ERK) signaling pathways.

Upregulation of miR-133b in cervical carcinoma cells strongly promotes both in vivo tumorigenesis and independent metastasis to the mouse lung (Qin et al., 2012).

Colorectal cancer

Note

Downregulation of miR-133b expression was significant in human colorectal cancer tissues compared with adjacent normal tissues.

Ectopic expression of miR-133b potently affected colorectal cancer cell proliferation and apoptosis in vitro and in vivo by direct targeting of the receptor tyrosine kinase MET (Hu et al., 2010). Overexpression of miR-145, miR-1, miR-146a, miR-576-5p, miR-126*, HS287, miR-28-5p, miR-143, miR-199b-5p, miR-199a-5p, miR-10b, miR-22, miR-133b, miR-145*, miR-199a, miR-133a, miR-125b and downregulation of miR-31 and HS170 were observed in brain-metastatic colorectal carcinomas (Li et al., 2012).

Prognosis

High expression of miR-185 and low expression of miR-133b were correlated with poor survival (p=0.001 and 0.028, respectively) and metastasis (p=0.007 and 0.036, respectively) in colorectal cancer. (Akçakaya et al., 2011).

Gastric cancer

Note

The most highly expressed miRNAs in non-tumorous tissues were miR-133b as well as miR-768-3p, miR-139-5p, miR-378, miR-31, miR-195, miR-497, compared to in gastric cancer tissues (Guo et al., 2009).

miR-133b was downregulated in high-grade gastrointestinal stromal tumors. Fascin-1 mRNA was upregulated in accordance with miR-133b downregulation in high-grade gastrointestinal stromal tumors; this result was consistent with a previous report showing that fascin-1 might be a direct target of miR-133b (Yamamoto et al., 2013). miR-133b targets FGFR1 and inhibits gastric cancer cell growth (Wen et al., 2013).

Lung cancer

Prognosis

MiR-133B had the lowest expression of miRNA in lung tumor tissue compared to adjacent uninvolved

tissue. Selective over-expression of miR-133B in adenocarcinoma (H2009) cell lines resulted in reduced expression of MCL-1 and BCL2L2. MiR-133B directly targets the 3'UTRs of both MCL-1 and BCL2L2. Lastly, over-expression of miR-133B induced apoptosis following gemcitabine exposure in these tumor cells (Crawford et al., 2009). miR-133b can inhibit cell growth of NSCLC through targeting EGFR and regulating its downstream signaling pathway (Liu et al., 2012).

Oncogenesis

Serum miR-206 and miR-133b were significantly up-regulated in the early stage of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone-induced lung carcinogenesis. miR-206 and miR-133b exhibited low-expression in lung cancer tissues (Wu et al., 2013).

Osteosarcoma

Note

A set of miRNAs, miR-1, miR-18a, miR-18b, miR-19b, miR-31, miR-126, miR-142-3p, miR-133b, miR-144, miR-195, miR-223, miR-451 and miR-497 was identified with an intermediate expression level in osteosarcoma clinical samples compared to osteoblasts and bone (Namløs et al., 2012).

Prostate cancer

Note

miR-133a and miR-133b are expressed at the detection limit in two hormone-insensitive prostate cancer cell lines, PC3 and DU145. Ectopic expression of miR-133 inhibited cell proliferation, migration and invasion in these cells, possibly by targeting EGFR (Tao et al., 2012). A significant lower expression of miR-1, miR-133b and miR-378* was observed in osteosarcomas with respect to control, and also in 31 high-grade osteosarcomas than in 25 low-grade and in metastatic versus non-metastatic patients. The expression of miR-1 and miR-133b may control cell proliferation and cell cycle through MET protein expression modulation (Novello et al., 2013).

Oncogenesis

miR-133b is directly up-regulated by androgen receptor, represses CDC2L5, PTPRK, RB1CC1, and CPNE3, and, is essential to prostate cancer cell survival (Mo et al., 2013).

Squamous cell carcinoma

Note

MiR133B expression was downregulated in laser microdissected cells of tongue squamous cell carcinoma (Wong et al., 2008b). MiR-133b as well as miR-145, miR-30a-3p, and miR-133a are downregulated in esophageal squamous cell carcinoma (Kano et al., 2010).

Oncogenesis

Tongue squamous cell carcinoma cell lines transfected with miR133a and miR-133b precursors displayed reduction in proliferation rate possibly through the downregulation of pyruvate kinase type M2 (Wong et al., 2008a). Gain-of-function analysis revealed that 3 transfectants (miR-145, miR-133a and miR-133b) inhibit cell proliferation and cell invasion in esophageal squamous cell carcinoma cells (Kano et al., 2010). miR-133b, was downregulated in esophageal squamous cell carcinoma tissue compared with the adjacent normal tissue. Bioinformatics analyses identified that miR-133b was found to be involved in invasion and metastasis of esophageal squamous cell carcinoma (Fu et al., 2013).

Muscular development

Note

miR-133b as well as miR-1, miR-133a, and miR-206 levels were found increased during late stages of human foetal muscle development. Increases in the expression levels of these miRNAs were proportional to the capacity of myoblasts to form myotubes. Changes in miRNA levels during human foetal development were accompanied by endogenous alterations in their known targets and also in their inducer, MyoD. Ectopic MyoD expression caused an induction of muscle cell differentiation in vitro, accompanied by an increase in the levels of miR-1, miR-133a, miR-133b and miR-206 (Koutsoulidou et al., 2011).

Myocardial hypertrophy and heart failure.

Note

MiR133B expression was downregulated in the heart obtained from idiopathic cardiomyopathy and ischemic patients (Sucharov et al., 2008).

Disease

Down-regulation of miR-133b induced an increase in cardiomyocyte size while over-expression of miR-133b dramatically reduced the cell size, suggesting that miR-133b may be a global regulator of cardiomyocyte hypertrophy (Sucharov et al., 2008).

Xiao et al. quantified the muscle-specific microRNA subtypes miR-133a and miR-133b, which can posttranscriptionally regulate and repress KvLQT1 protein expression without affecting mRNA expression (Xiao L et al., 2008). miR-1, miR-133a, miR-133b, and miR-208b were independently associated with high-sensitivity troponin T levels (all $P<0.001$) in plasma samples obtained on admission from 444 patients with acute coronary syndrome (Widera et al., 2011).

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